

**Public Health Goal for
DINOSEB
in Drinking Water**

Prepared by

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PREFACE

Drinking Water Public Health Goal of the Office of Environmental Health Hazard Assessment

This Public Health Goal (PHG) technical support document provides information on health effects from contaminants in drinking water. The PHG describes concentrations of contaminants at which adverse health effects would not be expected to occur, even over a lifetime of exposure. PHGs are developed for chemical contaminants based on the best available toxicological data in the scientific literature. These documents and the analyses contained in them provide estimates of the levels of contaminants in drinking water that would pose no significant health risk to individuals consuming the water on a daily basis over a lifetime.

The California Safe Drinking Water Act of 1996 (amended Health and Safety Code, Section 116365) requires the Office of Environmental Health Hazard Assessment (OEHHA) to adopt PHGs for contaminants in drinking water based exclusively on public health considerations. The Act requires OEHHA to adopt PHGs that meet the following criteria:

1. PHGs for acutely toxic substances shall be set at levels at which scientific evidence indicates that no known or anticipated adverse effects on health will occur, plus an adequate margin-of-safety.
2. PHGs for carcinogens or other substances which can cause chronic disease shall be based solely on health effects without regard to cost impacts and shall be set at levels which OEHHA has determined do not pose any significant risk to health.
3. To the extent the information is available, OEHHA shall consider possible synergistic effects resulting from exposure to two or more contaminants.
4. OEHHA shall consider the existence of groups in the population that are more susceptible to adverse effects of the contaminants than a normal healthy adult.
5. OEHHA shall consider the contaminant exposure and body burden levels that alter physiological function or structure in a manner that may significantly increase the risk of illness.
6. In cases of scientific ambiguity, OEHHA shall use criteria most protective of public health and shall incorporate uncertainty factors of noncarcinogenic substances for which scientific research indicates a safe dose-response threshold.
7. In cases where scientific evidence demonstrates that a safe dose-response threshold for a contaminant exists, then the PHG should be set at that threshold.
8. The PHG may be set at zero if necessary to satisfy the requirements listed above.
9. OEHHA shall consider exposure to contaminants in media other than drinking water, including food and air and the resulting body burden.
10. PHGs adopted by OEHHA shall be reviewed periodically and revised as necessary based on the availability of new scientific data.

PHGs adopted by OEHHA are for use by the California Department of Health Services (DHS) in establishing primary drinking water standards (State Maximum Contaminant Levels, or MCLs). Whereas PHGs are to be based solely on scientific and public health considerations without regard to economic cost considerations, drinking water standards adopted by DHS are to consider economic factors and technical feasibility. For this reason PHGs are only one part of the information used by DHS for establishing drinking water standards. PHGs established by

OEHHA exert no regulatory burden and represent only non-mandatory goals. By federal law, MCLs established by DHS must be at least as stringent as the federal MCL if one exists.

PHG documents are developed for technical assistance to DHS, but may also benefit federal, state and local public health officials. While the PHGs are calculated for single chemicals only, they may, if the information is available, address hazards associated with the interactions of contaminants in mixtures. Further, PHGs are derived for drinking water only and are not to be utilized as target levels for the contamination of environmental waters where additional concerns of bioaccumulation in fish and shellfish may pertain. Often environmental water contaminant criteria are more stringent than drinking water PHGs, to account for human exposures to a single chemical in multiple environmental media and from bioconcentration by plants and animals in the food chain.

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SUMMARY

A Public Health Goal (PHG) of 14 ppb is developed for dinoseb in drinking water. The PHG was calculated based on a lowest-observed-adverse-effect-level (LOAEL) of 1 mg/kg-day for cystic endometrial hyperplasia in female mice and hypospermatogenesis and atrophy/degeneration of the testes in male. This is the identical LOAEL used by the U.S. Environmental Protection Agency (U.S. EPA) to promulgate its Maximum Contaminant Level (MCL) for dinoseb of 0.007 mg/L (7 ppb). The federal MCL is based upon a Drinking Water Equivalent Level (DWEL) of 0.035 mg/L (rounded by U.S. EPA to 40 µg/L). No new scientific information was identified that would justify a change in the use of the LOAEL of 1 mg/kg-day. However, we identified three assumptions (body weight, relative source contribution and water consumption) previously used by U.S. EPA in the calculation of its MCL for which we have determined more appropriate values for use in PHG development based on current methodology which includes exposure contributions from inhalation and dermal contact with water-borne dinoseb, and the fact that dinoseb is no longer registered for use in California. Therefore, OEHHA calculates a PHG of 0.014 mg/L (14 ppb) for dinoseb in drinking water.

INTRODUCTION

Dinoseb (2-*sec*-butyl-4,6-dinitrophenol) and its salts are members of the dinitrophenol family of pesticides, a class of compounds used widely as herbicides and insecticides. Dinoseb was used extensively in the central valley of California and in the past it had been detected in ground water. Although dinoseb is rapidly degraded in surface water and soil via photolysis and microbial mechanisms, it is suspected to have a “long persistence” in ground water (Frank *et al.*, 1987; O'Neill *et al.*, 1989). No evidence was seen in the literature that dinoseb bioaccumulates. Nevertheless, the 1996 update of the California well inventory database (DPR, 1997) showed no detection of dinoseb in any of the 1,366 wells tested (3,564 total samples taken in 35 counties).

On October 14, 1986, U.S. EPA issued an emergency suspension order (U.S. EPA, 1986) for all agricultural products containing dinoseb, because the use of such products posed an “imminent hazard.” The specific concern was evidence that indicated exposure to dinoseb “poses a risk of birth defects, male sterility and acute toxicity to agricultural workers.” The suspension was based on occupational exposure considerations and on a developmental toxicity study in rabbits which showed frank teratogenicity with no maternal toxicity (Becker, 1986). Dinoseb was listed as known to the State of California to cause reproductive toxicity on January 1, 1989 (Proposition 65 list). Note that since there are no current registered uses of dinoseb in California, it is assumed that the compound is not used in agriculture and that the source of human exposure, if any, to this chemical would be via residues remaining in ground water.

TOXICOLOGY

For a more detailed discussion of the toxicology of dinoseb, refer to the 1992 Water Quality Criteria Document (U.S. EPA, 1992a) and the Canadian Water Quality Guidelines for Dinoseb (Environment Canada, 1991).

Toxicological Effects in Animals

Acute Toxicity

Oral LD₅₀ values for dinoseb range from 14 to 114 mg/kg in the rat, mouse, rabbit and guinea pig (Biggs *et al.*, 1964; Preache and Gibson, 1975a; Rowe *et al.*, 1966). Dermal LD₅₀ values in the rat fall in a similar range (67 to 134 mg/kg) (Noakes and Sanderson, 1969). Symptoms of poisoning include prostration, rapid respiration, hyperthermia and convulsions immediately preceding death. The precise mechanism of dinoseb toxicity is unknown. However, it is assumed that health effects following dinoseb exposure are due to the chemical's ability to uncouple oxidative phosphorylation. Other acute effects include depressed cellular immune responses, anorexia, methemoglobinemia in ruminants and cataract formation in ducklings and possibly in humans (Spencer, 1948; Froslic and Karlog, 1970; Froslic, 1976; Dandliker *et al.*, 1980; U.S. EPA, 1986).

Developmental and Reproductive Toxicity

Teratogenic effects of dinoseb in rats, mice and rabbits has been documented. Skeletal and neurological abnormalities have been reported in fetuses exposed to dinoseb at various stages of gestation (Preache and Gibson, 1975a,b; Kavlock *et al.*, 1985; Giavini *et al.*, 1986a,b). Generally, teratogenic effects are seen at higher doses (7.5 to 33.0 mg/kg-day) than are reproductive effects (1 to 22 mg/kg-day) (Environment Canada, 1991, Table 14).

Chronic Noncarcinogenic Effects

The most common effects of chronic exposure in rodents are reduced growth rates and weight loss. Long-term exposure (200 or more days) of mice and rats to sub-lethal doses of dinoseb (1 mg/kg-day) has also resulted in adverse effects on the testes, endometrium and decreased weight of offspring (Brown, 1981; Irvine and Armatage, 1981). Shorter exposures (3 to 70 days) to higher levels of dinoseb (7.5 to 22.2 mg/kg-day) have produced similar effects (Preache and Gibson, 1975a,b; Linder *et al.*, 1982; Giavini *et al.*, 1986a,b). Reduced fecundity (Preache and Gibson, 1975b) and decreased fetal survival (Spencer and Sing, 1982) have also been observed in rats and mice exposed to this compound.

Carcinogenicity

Two animal bioassays have been performed to evaluate the carcinogenic potential of dinoseb, one in the rat and one in the mouse. In the rat study (Hazelton, 1977), no increases in tumor incidences were seen at 1, 3 and 10 mg/kg-day dinoseb administered in the diet after 104 weeks. Mice, administered dinoseb in their diet for 100 weeks at the same levels, showed equivocal results (Brown, 1981). Statistically significant increases in combined adenomas and carcinomas were observed in the livers of female, but not male mice. No historical control information was reported and there was a lack of other hepatocellular changes commonly associated with hepatocarcinogenesis.

Genetic Toxicity

Results from mutagenesis assays have also been equivocal; some assays have been negative (Ames Assay, unscheduled DNA synthesis in human fibroblasts, recessive-lethal in *D. melanogaster*) and

some positive (DNA repair synthesis in several bacterial strains). Accordingly, U.S. EPA classified dinoseb as a class D carcinogen: not classifiable as to human carcinogenicity (U.S. EPA, 1992a).

Toxicological Effects in Humans

No relevant data on the toxicological effects in humans were available for review.

DOSE-RESPONSE ASSESSMENT

A lifetime health advisory (HA) of 7 ppb (0.007mg/L) was developed by U.S. EPA in August 1988 (U.S. EPA, 1988). This HA was based on a DWEL of 0.035 mg/L (rounded by U.S. EPA to 40 µg/L) and a relative source contribution (RSC) of 20%. The study upon which the U.S. EPA's reference dose (RfD) was based, which serves as the foundation for the DWEL, was a two-year dietary study in rats (Hazelton, 1977) which identified an LOAEL of 1 mg/kg-day. The endpoint of toxicity was a treatment-related decrease in mean thyroid weights in all dosed males.

In July 1992, U.S. EPA promulgated an MCL and a Maximum Contaminant Level Goal (MCLG) for dinoseb of 7 ppb (U.S. EPA, 1992b). The MCL and MCLG are identical to the HA which was based originally on the LOAEL identified in the Hazelton (1977) study. Upon additional review, however, U.S. EPA concluded that because only a limited number of animals were examined histopathologically in the Hazelton (1977) study, a more suitable study upon which to base the HA would be the Brown (1981) study. In this 100-week feeding study in the mouse, a LOAEL of 1 mg/kg-day was identified. The endpoints were cystic endometrial hyperplasia in female mice and hypospermatogenesis and atrophy/degeneration of the testes in male mice. This LOAEL is supported by two other studies: the Hazelton (1977) two-year dietary study in rats which identified a LOAEL of 1 mg/kg-day based on a treatment-related decrease in mean thyroid weights in all dosed males, and a three-generation reproductive study which demonstrated decreased fetal weights and a decrease in pup body weights at all doses (1, 3 and 10 mg/kg-day) (Irvine and Armitage, 1981). A LOAEL of 1 mg/kg-day can also be identified from the reproduction study.

Using the LOAEL of 1 mg/kg-day, U.S. EPA calculated its MCL (and MCLG) as follows:

Step 1: Determination of an RfD (in mg/kg-day)

$$\text{RfD} = \frac{\text{LOAEL}}{\text{UF}}$$

where,

LOAEL = Lowest-observable-adverse-effect-level (1 mg/kg-day).

UF = Uncertainty factor of 1,000 [10 for inter-species extrapolation, 10 for human variability and 10 for the use of a LOAEL instead of a no-observed-adverse-effect-level (NOAEL) to reflect a relatively severe endpoint].

Therefore,

$$\text{RfD} = \frac{1 \text{ mg / kg - day}}{1,000} = 0.001 \text{ mg/kg-day.}$$

Step 2: Determination of a DWEL (in mg/L)

$$\text{DWEL} = \frac{\text{RfD} \times \text{BW}}{\text{L/day}}$$

where,

RfD = Reference dose (0.001 mg/kg-day)

BW = Assumed body weight of an adult male (70 kg)

L/day = Assumed daily water consumption of an adult (2 L/day).

Therefore,

$$\text{DWEL} = \frac{(0.001 \text{ mg / kg - day})(70 \text{ kg})}{2 \text{ L / day}} = 0.035 \text{ mg/L.}$$

Step 3: Determination of the MCL (and MCLG) (in mg/L)

$$\text{MCL} = (\text{DWEL})(\text{RSC})$$

where,

DWEL = Drinking water equivalent level (0.035 mg/L)

RSC = Relative source contribution from water 20% (0.2).

Therefore,

$$\begin{aligned} \text{MCL} &= (0.035 \text{ mg/L})(0.2) \\ &= 0.007 \text{ mg/L} = 7 \text{ ppb.} \end{aligned}$$

CALCULATION OF PHG

The scientific literature since the 1992 promulgation of the MCL by U.S. EPA was reviewed for new information concerning the toxicity of dinoseb. No new sources of data which would influence the development of a PHG for this compound were identified¹. The study from which the MCL was derived (Brown, 1981) was reviewed and found to be adequate as a basis for the calculation of a PHG. Therefore, we concur with an LOAEL of 1 mg/kg-day, based on the combined effects of dinoseb on both the female and male reproductive systems in the rat, as the basis for the calculation for the PHG.

Calculation of a public health-protective concentration (C, in mg/L) for dinoseb in drinking water can be calculated according to the general formula for noncarcinogenic endpoints:

¹ A copy of the literature review is available upon request

$$C = \frac{(\text{LOAEL})(\text{BW})(\text{RSC})}{(\text{UF})(\text{L / day})} = \text{mg/L}$$

where,

LOAEL = Lowest-observed-adverse-effect-level (1 mg/kg-day)
 BW = Body weight for an adult female (60 kg)
 RSC = Relative source contribution of 80% (0.8)
 UF = Uncertainty factor of 1,000 (10-fold for inter-species extrapolation, 10-fold for human variability and 10-fold for the conversion of an LOAEL to an NOAEL for a relatively severe toxicity endpoint)
 L/day = Volume of water consumed per day for an adult (2 L/day).

Therefore,

$$\begin{aligned} C &= \frac{(1 \text{ mg / kg - day}) (60 \text{ kg})(0.8)}{(1,000) (2 \text{ L / day})} \\ &= 0.024 \text{ mg/L} = 24 \text{ ppb.} \end{aligned}$$

This calculation does not account for any possible contribution to a hypothetical dose of dinoseb from drinking water due to inhalation of volatilized material or via dermal exposure from activities such as showering. U.S. EPA has provided guidelines for incorporation of inhalation and dermal exposures from drinking water in the calculation of health advisory and DWEL values (U.S. EPA, 1989). Following their protocol, we have performed this analysis for the case of dinoseb. From this analysis, it is estimated (based on the physical-chemical characteristics of the chemical) that inhalation exposure would be approximately one-half that of oral exposure and dermal exposure would be approximately one-quarter that of oral exposure. Assuming oral exposure is due to drinking 2L/day of water, it follows that the inhalation exposure would be equivalent to drinking an additional 1L of water daily and the dermal exposure would be equivalent to drinking an additional 0.5L water per day. Accordingly, for the purposes of the PHG calculation the value of 3.5L water equivalents per day is used in place of 2L/day water consumption. The resulting PHG calculation is as follows:

$$\begin{aligned} C &= \frac{(1 \text{ mg / kg - day}) (60 \text{ kg})(0.8)}{(1,000) (3.5 \text{ L / day})} \\ &= 0.014 \text{ mg/L} = 14 \text{ ppb.} \end{aligned}$$

Thus OEHHA calculates a PHG of 0.014 mg/L (14 ppb) for dinoseb in drinking water.

RISK CHARACTERIZATION

The calculation used to derive the PHG for dinoseb is essentially the same calculation as that used by U.S. EPA to derive its MCL. The only differences are the use of an RSC of 80% (0.8) instead of 0.2, the use of the default value for adult female body weight (60 kg) instead of the default value of 70 kg for adult males which were used by U.S. EPA and the estimation of the exposure contribution from inhalation and dermal contact with water-borne dinoseb. Since dinoseb is no longer used agriculturally, it cannot enter the food supply or be distributed by air, therefore, the

value of 0.8 was selected because the sole source of exposure to dinoseb is expected to be from drinking water. Female body weight was used because the most sensitive toxicological endpoint of concern is adverse effects on female reproductive system. The calculation of the PHG accounts for contribution to a hypothetical exposure to dinoseb from drinking water due to inhalation of volatilized material or via dermal exposure from activities such as showering. U.S. EPA has provided guidelines for incorporation of inhalation and dermal exposures from drinking water in the calculation of health advisories and DWELs (U.S. EPA, 1989), but these exposures were not considered in the development of their MCLs or MCLGs. For dinoseb, OEHHA calculated a DWEL of 35 µg/L, assuming exposure to 2 L/day of water, contributed solely by the oral route and that there also could be 0.5 L “equivalents” by the dermal route and 1 L “equivalent” by the inhalation route. Nevertheless as a result of these assumptions, our calculated PHG of 14 ppb is greater than U.S. EPA’s MCL (and MCLG) of 7 ppb.

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